Three Cases of Carbon Tetrachloride Poisoning with One Fatality

It is thought to be worth while drawing attention once again to the hazards of carbon tetrachloride. This substance is often used for cleaning clothes, and when that is done in a small room lethal quantities may be inhaled, or it may accidentally be drunk, as is shown in Case 1.

Case 1

At about 8 p.m. a naval officer aged 30 drank about 14 ml. of carbon tetrachloride in lime juice. The carbon tetrachloride was kept for cleaning purposes in a gin-bottle from which the original label had not been removed. He realized immediately what had happened, and was advised by the medical officer on board to have his stomach washed out. This he declined to have done. He then drank a pint (570 ml.) of lime juice and water and vomited a few minutes later. Feeling better, he went on to a party, where he drank gin and beer. Next morning he got up feeling well at first, but three hours later vomiting and diarrhoea started. As this continued he was admitted to the Royal South Hants and Southampton Hospital on the second day after taking the carbon tetrachloride.

On admission he was jaundiced and his tongue was dry; his temperature was 99.2° F. (37.3° C.) and pulse 80; blood pressure 115/65. His liver was just palpable and was tender. His blood urea was 52 mg. per 100 ml., serum bilirubin 2.1 mg. per 100 ml., and serum thymol turbidity 1 unit. Measures were taken to combat fluid loss, including calcium gluconate, "casydrol," and glucose-saline intravenously. He continued to pass scanty urine, which contained red cells but no casts. His condition remained good for about 12 hours, but he then became drowsy and red in the face, following which cyanosis developed and he became delirious. The blood urea rose to 112 mg. per 100 ml. At 6 p.m. on the fourth day he was unconscious, and he died at 4.30 a.m. on the fifth day, about 104 hours after drinking the carbon tetrachloride.

The abnormal findings at necropsy were as follows: Externally there was a light jaundice. The region of the epiglottis showed fibrinous inflammation. The pyloric antrum of the stomach was congested, and the small intestine contained altered blood. The liver was large (2,970 g.) and showed passive congestion and yellow fatty change, with small areas of necrosis 2–4 mm. across. The spleen was large (390 g.), firm, and red. The kidneys were large (540 g.) and congested and their cortices were swollen. Small areas of haemorrhagic bronchopneumonia were found in the lungs, and the pleural surfaces showed numerous petechial haemorrhages posteriorly. The heart muscle was pale, and there were petechial haemorrhages, extensive at the base, and slight subendocardial haemorrhages.

On histological examination the liver was seen to be almost entirely destroyed. There was advanced centrilobular necrosis, with fatty degeneration of the cells in the less affected periphery of each lobule. The pulp of the spleen was engorged. The kidneys showed swelling and degeneration of the tubular epithelium. An occasional tubule was dilated and contained a hyaline cast. No changes were seen in the glomeruli. The changes in the liver were much more severe than those in the kidneys, and it was thought most probable that death was due to hepatic failure despite the normal serum thymol turbidity. Villela (1948) has, however, found this to be normal in 20–30% of rats and rabbits in which centrilobular necrosis had been induced by poisoning with carbon tetrachloride.

Case 2

A boiler-maker aged 23 was working in a confined space below a carbon tetrachloride spray which was being used for removing paint in a liner. It is not known for how long he was exposed to the spray. He collapsed, and when seen by the M.O. was unconscious and cold, with a poor colour and a thready pulse. Oxygen was given, and he recovered consciousness in 10 minutes, although still lacrimose, confused, cold, and photophobic. He was seen at the Royal South Hants and Southampton Hospital 50 minutes later, when his condition was quite normal. He was given calcium gluconate and casydrol by mouth. His urine was normal. Two days later his serum bilirubin was 0.15 mg. per 100 ml. and serum thymol turbidity 1 unit, and he was discharged. When seen three weeks later he was fit.

Case 3

This case was that of a girl aged 3 years. Her father had brought home some carbon tetrachloride for cleaning purposes in a Ministry of Food orange-juice bottle. The original brightly coloured label was still on the bottle, and no doubt the child thought she was helping herself to orange juice. At 6.30 p.m. she swallowed a little and almost immediately began to get drowsy, but her parents were not worried by this, as it was near her bedtime. Indeed, she was given some fish and chips, which had an immediate and most fortunate emetic effect. Her drowsiness increased, however, and she lost consciousness within three-quarters of an hour of taking the poison. She was admitted to hospital at 8.45 p.m. and given a stomach wash-out, the material from which contained a very small quantity of a volatile halogen compound. She did not regain consciousness until 12.50 the same night, having been unconscious for five and a half hours. Plenty of fluids, casinal in milk, and calcium gluconate, 20 gr. (1.3 g.), three times a day, were given, and she made an uninterrupted recovery. At no time did she show any evidence of renal or hepatic injury. The amount of carbon tetrachloride that was swallowed is not known, but her parents stated that it was not more than a thimbleful.

Comment

Sydney Smith (1936) states that alcohol given with this drug greatly increases its effect, and it seems probable that the alcohol consumed in Case 1 very soon after the drug increased its absorption in the intestines.

W. W. Woods (1946) reported three cases of fatal poisoning due to inhalation of "pyrene" fumes occurring aboard ship. In each case there was an interval of a few days between exposure and the development of symptoms. He described extensive kidney lesions resembling those seen in the "crush syndrome," but no such lesions were seen in my Case 1, which was subjected to a single larger dose than were his three cases. The typical centrilobular necrosis of the liver has often been described before, and, according to W. H. H. Andrews and B. G. Maegraith (1948), it is due to relative anoxia of the central portions of the lobules caused by swelling of the parenchymal cells of the liver.

Summary

A case of carbon tetrachloride poisoning is recorded in which death resulted from hepatic failure; reports are also given of two other cases which recovered.

Brief comment is made on similar cases reported in the literature.

My thanks are due to Dr. H. J. Bower and Dr. K. M. Robertson for permission to publish these cases, and to Dr. H. H. Gleave, who carried out the post-mortem examination on Case 1, for his help and criticism.

A. W. H. FOXELL, M.B., B.S.,
Senior Registrar, Pathological Department,
Royal South Hants and Southampton Hospital, Southampton.

REFERENCES

Andrews, W. H. H., and Maegraith, B. G. (1948). *Ann. trop. Med. Parasit.*, **42**, 95.
Smith, Sydney (1936). *Forensic Medicine*, 5th ed., p. 499. Churchill, London.
Villela, G. G. (1948). Quoted in *J. Amer. med. Ass.*, **136**, 1061.
Woods, W. W. (1946). *J. Path. Bact.*, **58**, 767.